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Arterial tortuosity in the femoropopliteal region during knee flexion: a magnetic resonance angiographic study

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ABSTRACT

Dynamic changes in curvature are expected in the femoropopliteal region during knee flexion. The location of the artery dorsal to the axis of movement implicates a relative length excess that may influence local morphology. To study arterial morphology in vivo, magnetic resonance angiography was performed in 22 healthy volunteers (aged 23–68 y). The curvature of the femoral vessels was studied and quantified in stretched and flexed positions. During knee flexion the vessel followed the movement of the leg and in the sagittal plane one curve was visible distal to the adductor hiatus. Three or more small curves were seen proximal to the knee joint in all volunteers. In the group aged under 30 y these minor curves were located proximal to the adductor hiatus as if the artery curls up in Hunter's canal. In the group aged over 45 y one or more curves were located distal to the adductor hiatus in the popliteal fossa. In volunteers aged 60 y and older some of these curves did not disappear during knee extension. In older individuals, natural elongation and loss of arterial elasticity will contribute to the formation of these curves. Impairment of the free gliding mechanism of the femoral vessels in the adductor canal could explain the differences in location of these minor curves between younger and older subjects. It is concluded that morphological changes in the femoral artery occur during knee flexion and that this tortuosity is age dependent. This may influence local haemodynamics and therefore possibly contribute to atherogenesis.

Key words: Vasculature; femoral artery; magnetic resonance angiography; atherogenesis.

INTRODUCTION

The distal part of the femoral artery is a well known predilection site for atherosclerosis. This phenomenon has been the subject of several investigations. Lindbom (1950) identified 2 principal sites for femoropopliteal occlusions, the main one being in the region of the adductor hiatus and the other in the popliteal area. This was confirmed by several other investigators (Mavor, 1956; Dunlop & Santos, 1957; Watt, 1965). Scholten et al. (1993) found 72% of the femoropopliteal occlusions at the level of the adductor canal hiatus.

Speculations have been made about the pathogenesis of atherosclerosis at this specific location. Besides general atherogenic causes, local anatomical factors such as surrounding structures, branches and local vessel morphology, are thought to play an important role. The abrupt transition in physical properties of the surrounding tissues is remarkable; in the adductor canal the femoral artery is surrounded by the firm muscles of the thigh whereas on leaving the canal the artery crosses the sharp edge of the aponeurosis of adductor magnus to enter the soft fatty tissue of the popliteal fossa. Watt (1965) suggested that unfavourable haemodynamic circumstances such as the S-shaped configuration of the femoral and popliteal arteries or frequent branching in this area contribute to the origin of atherosclerosis.

The morphology of the vessel will strongly influence local haemodynamics. With the knee extended the femoral artery shows a smooth elongated S shape. Smedby et al. (1993) described 2-dimensional tortuosity of femoral arteries affected by early athero-

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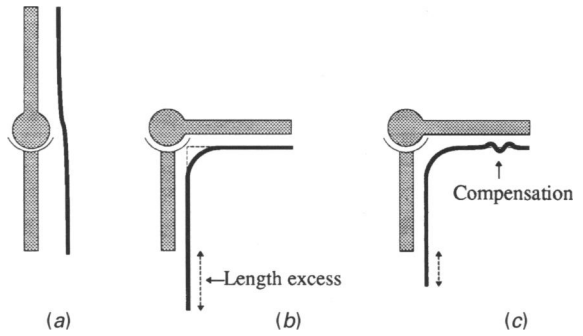

Fig. 1. Relative length excess of the femoral and popliteal artery during flexion of the leg, partly compensated by arterial tortuosity.

sclerotic lesions and suspected a relation between arterial tortuosity and atherosclerosis. Since the adductor region is close to the knee joint, flexion of the knee will certainly influence local morphology. Both Lindbom (1950) and Watt (1965) speculated on the influence of knee flexion on atherogenesis in the adductor and popliteal regions. They described possible changes in morphology of the artery during knee flexion, which undoubtedly have dramatic effects on local haemodynamics considering the close relation between haemodynamics and shape.

The position of the artery in the lower limb, behind the axis of movement, implicates excess of length in a flexed position (Fig. 1*a*, *b*). This is enhanced by the fact that the artery takes a short cut and bends smoothly instead of acutely. A smooth curve from thigh to calf will be promoted by the arterial bloodflow but limited by the space available and in turn will contribute to the already existing length excess.

There are two ways to deal with this length surplus: part of it will be absorbed in natural longitudinal elasticity of the vessel, while the remaining extra length leads to arterial tortuosity in the adductor canal and popliteal fossa.

The aim of this study is to determine how knee flexion influences 3-dimensional arterial morphology and how the relative arterial length excess that results from bending the knee is dealt with in healthy volunteers of different ages.

MATERIALS AND METHODS

To evaluate the morphology of the femoral artery in both the extended and flexed knee we used magnetic resonance angiography (MRA). Twenty-two healthy volunteers were divided into 4 different age-groups (see Table 1). No distinction was made between males and females.

MRA was performed with a 0.5 Tesla Philips Gyroscan T5, using a knee coil in extension and a wrap-around or body coil in flexion; 55 axial slices

Table. *Summary of the results*

	No.	Age	Sex	Radius of curve in sagittal plane (mm)	LE_1 (mm)	γ	Mean radius compensatory curves (mm)	Mean angle compensatory curves (°)	Compensation	Elasticity
Age-group 4	1	68	M	21	2.6	63	19.7	49.2	2.4	0.2
(> 61 y)	2	68	M	11	1.5	65	21.8	45	2.6	−1.1
Age-group 3	3	56	M	29	4.1	65	49.3	44.7	3	1.1
(46–60 y)	4	55	F	33	11.5	85	30.8	38.2	2.5	9
	5	52	M	32	6.3	72	58	33	1.8	4.5
	6	51	M	25	9.1	86	33	52	3.3	5.8
	7	47	M	16.5	2.9	70	82	34.7	2.5	0.4
Age-group 2	8	44	M	22.5	4.4	72	71.3	34.7	1.7	2.7
(31–45 y)	9	36	M	42	6.9	68	72	29	1.4	5.5
	10	35	M	84	13.6	68	69	33.3	1.7	11.9
	11	34	M	45	8.9	72	116.3	26.3	1.3	7.6
	12	31	M	127.5	11.7	57	51	34.3	1.4	10.3
Age-group 1	13	30	M	40	12.3	82	93	32.5	1.2	11.1
(20–30 y)	14	29	M	115	6.5	49	75	33	1.2	5.3
	15	29	M	75	16.1	74	67	34	1.7	14.4
	16	29	F	75	16.1	74	46.3	36.7	1.5	14.6
	17	23	M	73	19.7	79	62.7	34	1.5	18.2
	18	28	F	81	11.3	65	130.3	28	1.1	10.2
	19	28	M	60	12.4	73	167.3	24	1	11.4
	20	25	M	178	12.1	52	53.7	30.7	1	11.1
	21	24	F	41	11.1	79	58.7	31.3	1.2	9.9
	22	23	F	126.3	20.5	68	96	19.3	0.5	20

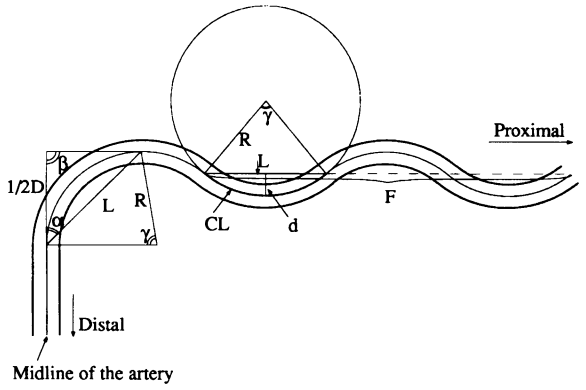

Fig. 2. Summary of all calculated parameters.

were obtained using a cardiac triggered turbo field echo inflow sequence. Scan parameters were FOV 30 cm, slice thickness 4 mm, no gap, TR 13 ms, TE 6 ms, inversion delay 250 ms, trigger delay 253 ms, flip angle 70°, matrix 256 × 128, one excitation, first-order flow compensation and a caudal presaturation slab. Three-dimensional reconstructions were made by using a maximum intensity projection technique, showing 16 different projections of a 22 cm long trajectory of the femoral and popliteal arteries, rotating around the vertical axis from 0 to 180°.

Although there is no accepted definition for tortuosity, it usually indicates the turns and twists in the course of an artery between one point and another. Although Smedby et al. (1993) described several ways to assess arterial tortuosity they used their own definition and mathematical methods. Unfortunately they only had 2-dimensional data to evaluate.

To evaluate arterial tortuosity in the healthy volunteers participating in this study, a series of geometric calculations were performed. Our main interest was to investigate the way in which the femoral artery copes with length excess as a result of knee flexion. Flexion of the knee causes separate parts of the femoral and popliteal artery to twist in two ways. One part incorporates the main curve in the sagittal plane that follows the direction of the leg and contributes to the length excess, the other involves small curves compensating for the length excess.

First the length excess caused by smooth bending of the artery was calculated. This length excess was termed LE_1. The length excess caused by the location of the artery behind the axis of movement could not be deduced from our data but was considered to be nonvariable. This length excess was termed LE_2. The total length excess is the sum of LE_1 and LE_2 (Fig. 2). Secondly, the length gained by arterial tortuosity was calculated. This part compensates partly for the total length excess. This was called compensation (C).

As stated in the Introduction the length excess not compensated for by the tortuosity of the artery will be absorbed in arterial elasticity. The elasticity itself cannot be measured with our data but can be considered proportional to the length excess LE_1 minus the compensation C. Details of all calculations are given in the Appendix.

RESULTS

In all volunteers the femoral and popliteal arteries became tortuous during knee flexion. Naturally the artery follows the movements of the leg and one curve becomes visible in the sagittal plane. The radius of this curve decreases with increasing age (Fig. 3). Consequently the length excess calculated in formula 4 (see Appendix) diminishes significantly with age

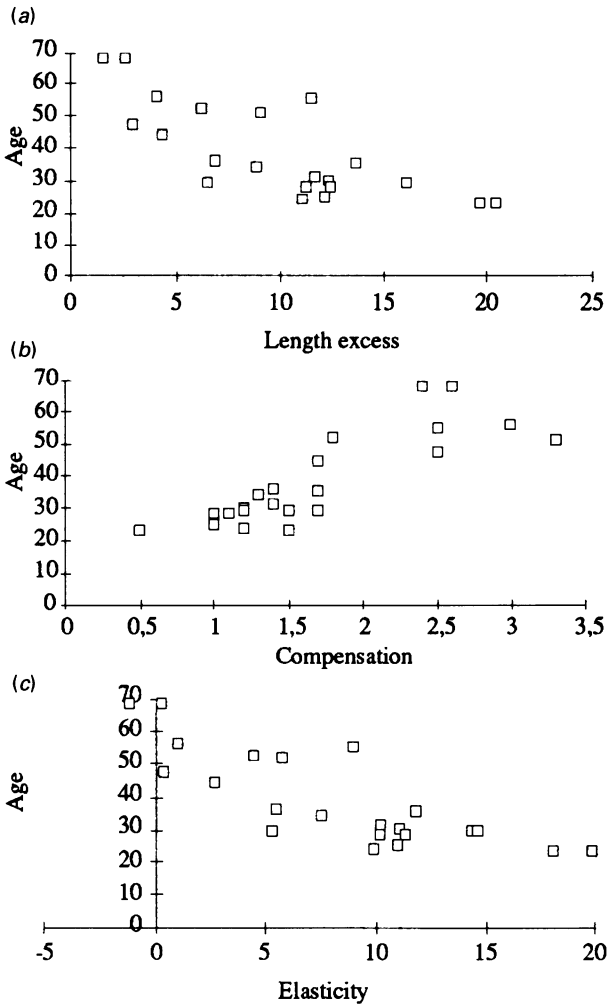

Fig. 3. (*a*) Length excess vs age. Correlation coefficient: −0.757 ($P < 0.0001$). (*b*) Compensation vs age. Correlation coefficient: 0.832 ($P < 0.0001$). (*c*) Elasticity vs age. Correlation coefficient: −0.803 ($P < 0.0001$).

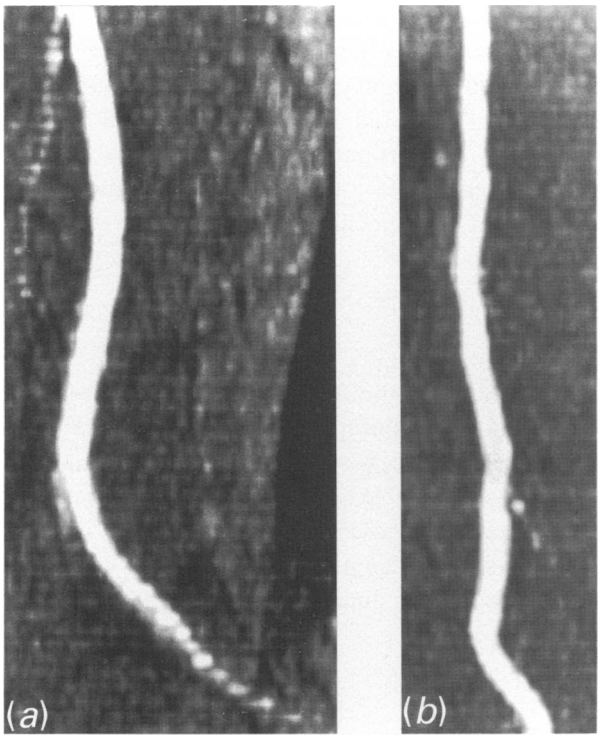

Fig. 4. (*a*) Lateral view of the femoral and popliteal arteries in a 30-y-old subject during 80° flexion. Note the smooth curve from thigh to calf. (*b*) Lateral view of the femoral and popliteal arteries in a 68-y-old subject during 80° flexion. Note the sharp acute curve from thigh to calf.

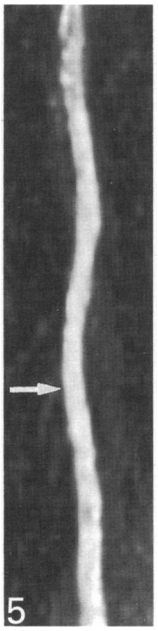

Fig. 5. Coronal view of the femoral and popliteal artery in a 30-y-old subject during 80° flexion. Three small consecutive curves are seen, all located in the adductor canal. The level of the hiatus of the adductor canal is indicated by the arrow.

(Fig. 4*a*). This means that in older individuals the artery makes more efficient use of the space available.

Besides this main curve, all subjects showed 3 or more smaller curves in the femoral and popliteal artery proximal to the knee joint. Formation of these curves seems to be age-dependent. In age-group I (< 30 y) all these curves were located proximal to the adductor canal hiatus as if the artery became concertinaed in the adductor canal. They were only visible in the coronal plane (Fig. 5). In the age-groups with older subjects a number of these curves were located distal to the hiatus of the adductor canal. Closer inspection revealed that curves located outside the adductor canal were apparent in the coronal as well as in the sagittal plane (Fig. 6). This implies a nonplanar configuration of the artery.

The mean radius of all 67 calculated small curves was 63 mm (20–167 mm) and showed no significant relation with age. The mean angle of all 67 curves was 33.5° (19–49°) and demonstrated a significant positive correlation with age. The compensation (C) made by the artery in meandering through the adductor canal and popliteal fossa is directly influenced by the radius and the angle of these small curves (see formulae A 5 and A 6 in the Appendix). Again a significant positive correlation with age was established (Fig. 4*b*). The calculated equivalent of the longitudinal arterial elasticity showed a significant negative correlation with age (Fig. 4*c*).

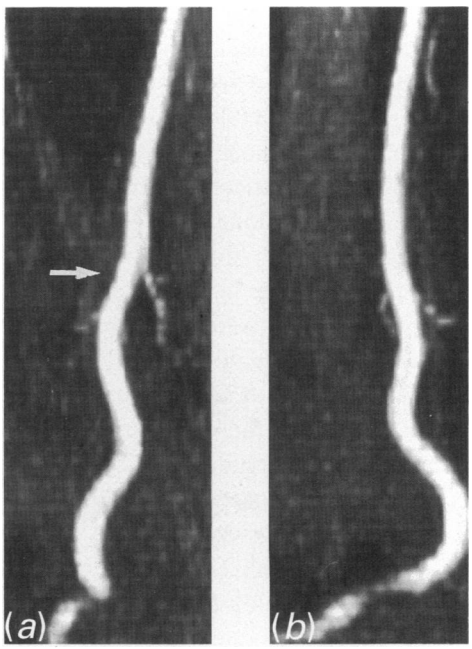

Fig. 6. Coronal view of the femoral and popliteal artery in a 51-y-old subject during 80° flexion (*a*). Note the small consecutive curves, located distal to the hiatus of the adductor canal. The level of the hiatus is indicated by the arrow. Lateral view of the same subject (*b*). Note the small consecutive curves in this plane, perpendicular to the plane in (*a*).

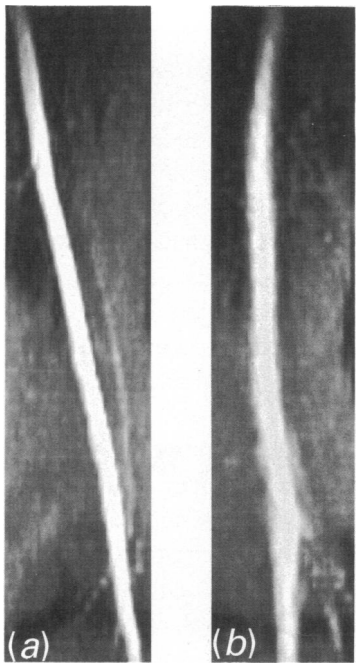

Fig. 7. Coronal (*a*) and lateral (*b*) views of an extended leg in a 21-y-old subject. The artery has regained its normal physiological morphology and the small curves have disappeared.

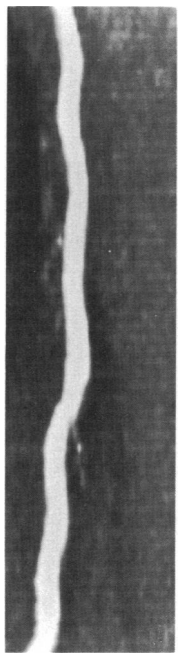

Fig. 8. Coronal view of an extended leg of a 68-y-old subject. The artery has not regained the smooth elongated S shape as in Figure 6*a* and *b*, some small curves remaining visible.

In all 15 volunteers aged under 45 y the artery regained its smooth elongated S shape when the knee was extended (Fig. 7). In the older subjects, however, in 3 of 5 cases, the original S shape, as seen in the younger subjects, was not completely restored in extension. Two or more small curves remained visible, superimposed on the elongated S shape (Fig. 7). No apparent differences were established between males and females.

DISCUSSION

As might be expected, the arterial length excess that results from bending the knee causes the morphology of the femoral and popliteal artery to change dramatically. The length excess that is not compensated by longitudinal elasticity causes the artery to concertina in the adductor canal and popliteal fossa. The differences in this tortuosity between older and younger individuals are influenced by 3 factors: (1) arterial elongation; (2) reduction of longitudinal arterial elasticity; and (3) impaired gliding of the femoral vessels in the adductor canal. The first 2 factors explain most of the differences. Arteries tend to elongate with increasing age (Chilvers et al. 1974), adding to the already existing length excess when the knee joint is flexed. Length excess should be compensated for. Considering the results of this investigation the main compensating mechanism will be arterial elasticity (see Table). Learoyd & Taylor (1966) found that the longitudinal and circumferential viscoelastic properties of arteries diminish with age, especially in the peripheral vessels. Mozersky et al. (1972) found a decrease of circumferential elasticity with increasing age in the femoral artery. Consequently, the demand for arterial tortuosity will be greater with increasing age, despite the vessel's more effective use of the space available by making an acute angle instead of a smooth curve.

Gliding of the femoral vessels in the adductor canal in both longitudinal and transverse directions was described by de Souza et al. (1984). In his opinion, free movements of the vessel in the adductor canal during muscle contraction are necessary to avoid traction on the vessel wall. According to Novotny (1950) this gliding mechanism is impaired in the elderly, due to perivascular fibrosis. Splitting of the adductor canal and stripping of the perivascular tissues was considered to be advantageous for patients suffering from stenosis or occlusion of the artery at this level, because this measure improved the local collateral circulation. Impairment of the gliding mechanism influences the location of compensatory curves during knee flexion. The limited retraction of the artery in the adductor canal promotes the location of curves in the popliteal fossa in older subjects.

Compensatory curves located in the adductor canal are found in the coronal plane because they are

restricted to the shape of the canal which is more or less rectangular, with the length of the rectangle in the coronal plane. Curves located distal to the hiatus of the adductor canal in the popliteal fossa are not restricted by the boundaries of the canal. The artery's freedom of movement accounts for the nonplanar configuration in the popliteal fossa.

Vernon et al. (1987) investigated 6 lower limbs post mortem during knee flexion with angiography in the sagittal plane and calculated that if the popliteal artery made a harmonious curve the arterial axis would shorten by about 25% at 90° flexion. Typical curves in the upper part of the artery compensate for the arterial length excess. These morphological changes take place between 2 fixed points, the adductor canal hiatus proximally and the origin of the anterior tibial artery distally. This contradicts de Souza's theory (1984) which described a gliding mechanism of the femoral vessels in both longitudinal and transverse directions. Zocholl et al. (1990) investigated 25 patients with digital subtraction angiography while the knee joint was flexed and shared Vernon's view on the existence of these 2 fixation points. In one young patient a single harmonious curve was observed whereas the popliteal artery showed a number of curves in 24 older patients. According to Zocholl and colleagues, loss of arterial elasticity with increasing age was considered to be the cause. In this study no significant relation was established between the number of curves and age; however, no measurements as to the radius or angle of the curves were performed.

Our results contradict the opinion that the popliteal artery is fixed at the hiatus of the adductor canal. This may be the case in older people with severe perivascular fibrosis, but it is definitely not true in young healthy individuals.

The importance of haemodynamics in atherogenesis has received increasing attention. It was originally postulated by Caro et al. (1971) and Caro (1977) and subsequently elaborated by Zarins et al. (1983), McMillan (1985) and several others. Atherosclerotic lesions tend to develop at sites where wall shear stresses are expected to be low. These shear stresses are determined by local haemodynamic factors, for instance blood flow velocity and the geometry of the artery. It must be admitted, however, that the large majority of atherosclerotic plaques develop at sites where no mechanical explanation is obvious.

Natural elongation and loss of arterial elasticity also account for the fact that with age the artery loses the smooth elongated S shape on extension. Considering the close relation between form and flow this implies more permanent disturbances of bloodflow and possibly a fixed pattern of low wall shear stresses which may initiate atherosclerosis. It is clear that many plaques develop at sites where the relation with haemodynamics is not so evident. This might also have been said of the femoral artery until our study demonstrated the possible relation between haemodynamics and atherosclerosis. From these observations it could be speculated that frequent movements of the leg and consequently frequent changes of arterial shape and the redistribution of low wall shear stresses will be factors that delay the atherosclerotic process.

From a clinical perspective, these findings may have considerable consequences. Kimball et al. (1990) found that tortuosity in coronary arteries adversely influences the results of angioplasty. Another recanalisation technique is the use of endovascular stents. According to Zocholl et al. (1990) the implantation of stents will not improve the artery's elasticity. The arterial length excess when the knee joint is flexed, in combination with the different physical properties of the stent material and the artery, will cause excessive kinking at the transition between the stented and nonstented vessel segments. This will lead to unfavourable haemodynamic circumstances which may contribute to restenosis.

It might be beneficial for patients who undergo recanalisation of any kind in the femoropopliteal area to avoid flexion of the knee joint for long consecutive periods in order to contribute to more favourable haemodynamic circumstances in the recanalised artery.

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APPENDIX

For every curve observed in MR angiography reconstructions the radius R was calculated (mm), as if the curve were part of a circle. To calculate the radius of a curve the formula

$$R = \frac{d^2 + (1/2L)^2}{2d} \qquad \text{(A 1)}$$

is used, where L is the shortest distance between the endpoints of the curve and d is the perpendicular distance from the middle of L to the curve. These 2 parameters can be measured on the MR angiograms using the midline of the vessel (Fig. 2). In each case, the plane in which the curve was maximal, was used to make the measurements.

With the radius, the curve length CL (mm) was calculated

$$\mathrm{CL} = 2\pi R^{*}(\gamma/360) \qquad \text{(A 2)}$$

where γ is the angle the curve makes in degrees (Fig. 2).

For the major curve in the sagittal plane, caused by the angle of flexion, the distance (D) the artery would cover if it made an acute angle instead of a length consuming smooth curve was calculated (mm)

$$1/2D = \frac{1/2L}{\cos\alpha} \qquad \text{(A 3)}$$

where $\alpha(^\circ) = 1/2(180-\beta)$ and $\beta(^\circ) = 180-\gamma$ (Fig. 2).

The length excess LE_1 (mm) that results from a smooth short cut as opposed to an acute angle is defined as

$$\mathrm{LE}_1 = D - \mathrm{CL}. \qquad \text{(A 4)}$$

The length excess LE_2 caused by the location of the artery behind the axis of movement was not calculated. If the angle of flexion is 90°, LE_2 equals twice the distance between the midline of the artery and the axis of movement. These parameters could not be deduced from the midline of the artery and the axis of movement. These parameters could not be deduced from the MR angiograms.

The distance F (mm) the artery would cover if it were a straight tube instead of a tortuous one, was measured (Fig. 2). This distance was deducted from the sum of all curve lengths (ΣCL). The outcome was the distance compensated for by meandering of the artery. We called this compensation C (mm) (Fig. 2).

$$C = \Sigma\mathrm{CL} - F. \qquad \text{(A 5)}$$

For every individual the compensation C calculated above was deducted from the calculated length excess LE_1. The result represents the length excess not compensated for by arterial tortuosity but by arterial elasticity. If the length excess LE_2 is considered a nonvariable, which seems valid because the distance between the midline of the artery and the axis of movement is not expected to show large variation with increasing age, it will be proportional to longitudinal arterial elasticity AE.

$$\mathrm{AE} \sim \mathrm{LE}_1 - \mathrm{C}.$$